THE TAMILNADU Dr. M.G.R. MEDICAL UNIVERSITY
CHENNAI – TAMILNADU.

DISSERTATION
ON

EEG CORRELATION WITH CT SCAN BRAIN FINDING IN PATIENTS WITH STROKE

SUBMITTED FOR M.D. DEGREE EXAMINATION
BRANCH I
(GENERAL MEDICINE)

EXAMINATION
IN
MARCH – 2007

THANJAVUR MEDICAL COLLEGE
THANJAVUR

CERTIFICATE

This is to certify that this dissertation entitled " **EEG CORRELATION WITH CT SCAN BRAIN FINDING IN PATIENTS WITH STROKE"** is the bonafide record work done by **Dr. K. KALYANARAMAN,** submitted as partial fulfillment for the requirements of M.D. Degree Examinations, General Medicine (Branch I) to be held in March 2007.

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ACKNOWLEDGEMENT

I am greatly indebted to my unit chief and beloved teacher **PROFESSOR OF MEDICINE AND THERAPEUTICS DR. S. BALAKRISHNAN, M.D.,** who inspired, encouraged and guided me in every step of this study.

I express my sincere gratitude to **PROFESSOR AND HEAD OF THE DEPARTMENT OF MEDICINE PROF. DR. K. GANDHI, M.D.,** for his valuable support and guidance in preparing this dissertation

I am extremely grateful to **THE DEAN, THANJAVUR MEDICAL COLLEGE** for granting me permission to do this dissertation work in Thanjavur Medical College Hospital, Thanjavur.

I am greatly indebted to Neurology Chief and beloved teacher **PROF. DR. P.S. GANESAN, M.D., D.M., (NEUROLOGY)** who guided me in this study.

I would like to gratefully acknowledge the help rendered by **Dr. M. THANGARAJ, M.D., D.M.** (Neurology) and **Dr. ANGURAJ, M.D., D.M.,** (Neurology) who was kind enough to guide me throughout this study.

I am thankful to my beloved professor Dr. **N. JEEVA,** M.D., professor of Medicine, **Dr. S. MUTHUKUMAR, M.D.,** and Assistant Professors **Dr**. **K. RAM SEKAR**, **M.D., Dr. A. SRINIVASAN, M.D.,** of my unit for their guidance and help throughout this study.

I thank the Radiology and Bio chemistry Department for their help in investigation aspects.

I express my gratitude to all the patients who participated in this study.

CONTENTS

INTRODUCTION

Stroke is defined as abrupt onset of focal neurological deficit due to a focal vascular cause[53]. Stroke is the third most common cause of mortality and morbidity[50].

EEG can be used as a diagnostic tool in patient with seizures. The importance of EEG in the evaluation of stroke in often ignored. In 30-50% of patient with an acute ischemic stroke, the CT Scan shows no abnormality. CT Scan takes 1to 5 days to detect infarct.

Early diagnosis and treatment is necessary in patients with stroke. MRI is superior to CT Scan in detecting small lesions. In most of the centers MRI is not available, due to its high cost. In most of the centers EEG is available. Hence in these centers EEG changes can be used as a second line investigation to localize cerebral ischemia.

AIM OF THE STUDY

1. To assess the role of EEG in patients with acute cerebrovascular accident and its correlation with CT Scan.
2. To study EEG changes in cortical and sub cortical infarct.
3. To study the effects of stroke in the opposite hemisphere.

REVIEW OF LITERATURE

The electroencephalogram has made a remarkable contribution to the diagnosis of cerebrovascular lesion after its discovery by HANS BERGER In an acute stroke a massive and highly impressive EEG focus may be present before computerised tomography can demonstrate the lesion.[10] MRI has been found to be even more precise and more informative than CT in the morphological evaluation of stroke; with MRI lesion can be visualised within about 2 to 6 hours after the vascular accident, whereas CT Scan demonstration usually requires 1 to 5 days before positive results are obtained.

The EEG is an indicator of tissue breakdown before the structure shows its sufferring [11]. Considerable localising value has been ascribed to local depression of the background activity while more extensive depression is regarded as somewhat less informative [14] The preservation of fast background activity in subacute forms of middle cerebral artery ischemia is indicative of considerable neuronal survival in the infarcted zone and hence indicative of a good prognosis[13, 14, 20]. The physiological factors play a major role in the pathogenesis of EEG abnormalities in cerebral infarct,thus accounting for the discrepancies between the EEG parameters and CTScan findings.It is suggested that EEG should be included routinely in the investigation of cerebral infarct [7]

EEG has a sensitivity of 77 % and specificity of 75 %[8].EEG findings can also provide physiological data , in that the cortical lesions are likely to be due to embolism, while the subcortical lesion are more likely to be due to the pathological process of intracerebral blood vessels and the lesion of the watershed territories to hemodynamic phenomena[8] .

Electroencepahalography is particularly useful following stroke, if the initial CT excludes hemorrhages but does not detect infarction. In conjunction with clinical details, EEG can then be used to indicate the likely pathophysiological mechanism of infarction[9]. Clinically important focal electroencephalographic abnormalities may occur in the absence of CT lesions, and clinicians must understand the implications of such dissociation[10]. Continuous focal abnormalities correlated with large lesions, mass effect and altered state of consciousness. Reactive focal abnormalities were associated with small lesions and the abscence of mass effect. Ipsilateral background abnormalities correlated with lesion size and mass effect. Attenuation of ipsilateral background activity was more important than irregularity .Abnormal background activity contra lateral to the side of lesion was associated with alteration of consciousness [11].

EEG CHANGES IN VARIOUS FORMS OF STROKE

STEM OF MIDDLE CEREBRAL ARTERY THROMBOSIS

In middle cerebral artery thrombosis the EEG shows pronounced irregularity and very slow delta activity ipsilaterally with frontotemporal maximum[13] [14]. Traces of sharp activity may be present over affected areas. Marked depression of background activity may be observed in severe strokes [19.] Focal intermittent rhythmical delta runs are seen along with local or diffuse polymorphic slowing [18]. Background abnormalities are of more prognostic value than focal slowing [20] . The EEG reflects a variety of stroke complications and provides valuable assessment of the patients condition [21].

Preservation of background activity is a prognostically favourable sign for patients recovering from stroke [23] . In hemiplegic stroke augmentation of rolandic mu rhythm may develop over affected side[24] . The EEG can be used for continuous monitoring after stroke[25 .]

EEG IN OCCLUSION OF DEEP BRANCHES OF MIDDLE CEREBRAL ARTERY

EEG shows little or no focal slowing;occasionally ipsilateral slowing is due to ischemia in superficial branches of the middle cerebral artery [14] .

EEG IN THALAMIC INFARCT

There are no EEG changes

HEMORRHAGE INVOLVING THE CAPSULAR REGION

EEG changes may not be quite as impressive as the clinical picture,probably due to preservation of cortical garland. Delta activity may be present maximally over frontotemporal region [26].

ANTERIOR CEREBRAL ARTERY STROKE

Ipsilateral frontal delta activity is a typical EEG finding; delta activity may be rhythmical intermittent character [14]

PERFORATING BRANCHES OF ANTERIR CEREBRAL ARTERY

EEG is characterized by frontal intermittent rhythmical delta activity [14].

POSTERIOR CEREBRAL ARTERY

EEG shows accentuated delta activity over the ipsilateral parieto occipital region [27]. EEG in thrombosis of external geniculate artery of posterior cerebral artery shows varying degrees of slow activity which can be quite impressive in thalamic leads[28].

EEG IN EMBOLIC STROKE

EEG shows spike and sharp waves in a large population of embolic strokes[29]. In general EEG can not assist in differentiating embolic strokes from thrombotic strokes.

ELECTROENCEPHALOGRAPHY

The electroencephalogram (EEG) is the depiction of the electrical activity occurring at the surface of the brain. This activity appears on the screen of the EEG machine as waveforms of varying frequency and amplitude measured in voltage.

EEG waveforms are generally classified according to their frequency, amplitude, and shape, as well as the sites on the scalp at which they are recorded. The most familiar classification uses EEG waveform frequency (eg, alpha, beta, theta). Information about waveform frequency and shape is combined with the age of the patient, state of alertness or sleep, and head site to determine significance. Normal EEG waveforms, like many kinds of waveforms, are defined and described by their frequency, amplitude, and location.

- Frequency (Hertz, Hz) is a key characteristic used to define normal or abnormal EEG rhythms.

- Most waves of 7.5 Hz and higher frequencies are normal findings in the EEG of an awake adult. Waves with a frequency of 7 Hz or less often are classified as abnormal in awake adults, although they normally can be seen in children or in adults who are asleep. In certain situations, EEG waveforms of an appropriate frequency for age and state of alertness are considered abnormal, because they occur at an inappropriate scalp location or demonstrate irregularities in rhythmicity or amplitude.

- Some waves are recognized by their shape, scalp location or distribution, and symmetry. Certain patterns are normal at specific ages or states of alertness and sleep.

- The morphology of a wave may resemble specific shapes, such as vertex (V) waves seen over the vertex of the scalp in stage 2 sleep or triphasic waves that occur in the setting of various encephalopathies.

- The frequencies of most brain waves range from are 0.5-500 Hz. However, the following categories of frequencies are the most clinically relevant:

- Alpha waves - 8-13 Hz

- Beta waves - Greater than 13 Hz

- Theta waves - 3.5-7.5 Hz
- Delta waves - 3 Hz or less

THE NEURAL BASIS OF THE EEG

NEUROPHYSIOLOGICAL MECHANISM

Many mechanisms have been proposed to account for the repetitive slow potentials that can be recorded from the surface of the brain or from the scalp. An adequate explanation depends on knowledge of the following.

1. The identities of the cells generating the electrical potentials.
2. They type of electrical potential involved.
3. The neural network determining the frequency and variation in amplitude of the potentials.
4. The neural mechanisms responsible for modifying the potentials when the excitability of the brain is altered.

Since Berger's original description of the EEG it has generally been assumed that the EEG results from the electrical activity of cells in the cerebral cortex. The following are neurophysiological basis of the EEG.

1. The repetitive waves which can be recorded from the surface of the brain or from the scalp are summated synaptic potentials generated by the pyramidal cells in the cerebral cortex.

2. The synaptic potentitals are the responses of the cortical cells to rhythmic discharges from thalamic nuclei.

3. The frequencies and sizes of the thalamic discharges (and hence of the cortical potentials) are determined by the special arrangements of excitatory and inhibitory interconnections among thalamic cells.

4. During 'activation', inputs from the reticular formation abolish the rhythmic discharges in the thalamic nuclei and cause the cortical potentials to become desynchronized.

CELLS GENERATING EEG POTENTIALS

The three types of cell found in the cerebral cortex have already been described and classitied as stellate, spinate or pyramidal. Among these cells, only the pyramidal cells contribute to slow waves which is recorded over the surface of the cortex. EEG recording electrodes are large and distant from the cell potential generators, they will only detect the summed activities of large numbers of adjacent dipoles in the same axis. The pyramidal cells are the only ones uniformly orientated (perpendicular

to the cortical surface) and with dendrites long enough to form effective dipoles[40].

ELECTRICAL ACTIVITY RESPONSIBLE FOR CORTICAL SLOW WAVES

EEG potentials result from the flow of current in the extracellular fluid surrounding nerve cells. The nerve cell membranes exhibit several types of activity such as.

1. Propagating dendritic potentials
2. Non – propagating somatic and dentritic postsynaptic potentials (excitatory and inhibitory)
3. Action potentials of axon hillock and soma.
4. Action potentials of axon.

It is unlikely that activity in axons or fine dendrites can contribute substantially to surface waves. The reason in both cases is that the narrow diameters of the structures impose considerable internal resistance to the flow of ionic current. Hence the potentials developed extra cellularly are small. Compelling evidence says that axons are relatively unimportant in the genesis of the EEG based on animal experiment[41]. Maximal stimulation of the nerve evoked propagated action potentials which were barely detectable by large electrodes on the surface of the cortex.

In contrast to the axons and find dendrites, the extra cellular action potential developed during the soma (and axon hillock) is relatively large. However, the duration of the soma action potential ‘ spike’ is brief (for example, 0.3 ms) and perfectly synchronous firing amongst adjacent cells is unlikely. Consequently there is little chance of the extracellular potentials summing effectively at the surface of the cortex. In animal experiments it is still possible to record spontaneous cortical waves when the anaesthetic dose is large enough to prevent the initiation of action potentials in cortical cells[42]. For these reasons it is unlikely that soma action potentials make an important contribution to surface waves. All the available evidence suggests that these waves result from excitatory or inhibitory postsynaptic potentials developed by the soma and larger dendrites. The low internal resistances of these structures ensure that the extra cellular potentials will be correspondingly large and the slow time courses of the postsynaptic potentials permits summation with those of other cells. It is also possible that propagating dendritic potentials will contribute to the surface waves especially when the larger dendrites are invaded. In conclusion, intracellur recordings strongly favour postsynaptic potentials as being responsible for surface waves. It has been found in many cortical neurons that repetitive postsynaptic potentials exhibit a close time relationship with potentitals recorded on the surface of the overlying cortex[43] .

LOCATION OF THE EEG PACEMAKER

The question next arises as to whether the repetitive postsynaptic potentitals in the pyramidal cells are evoked by discharges from other cortical cells or from a subcortical pacemaker. One experimental approach has been to study the electrical activity in a slab of feline cortex which has been neurally isolated by cutting fibre connections to all other regions of the brain. Such unanaesthetized slabs remain viable since they receive an adequate blood supply through pial vessels and they can still respond to direct electrical stimulation. Unfortunately, conflicting observations have been made on such slabs. Although Burns recorded no spontaneous potentials whatsoever, other workers have reported the occurrence of backgound activity, including spindles[44] . From this last work it would appear that the cortex does contain neural networks which can generate spontaneous rhythms. On the other hand, two observations suggest that the cortical networks are normally driven by an external pacemaker. Firstly, it has already been seen that spontaneous waves can still be recorded when cortical impulse activity, and therefore intracortical driving, has been abolished by anaesthesia. Secondly, if the thalamus is removed on one side by suction it is found that spontaneous spindles are abolished for several hours in the ipsilateral hemisphere[44] .

These last observations strongly suggest that some other part of the brain is normally responsible for driving the slow wave activity in the cortex. Bremer et al showed by a simple but very meaningful experiment that

the site of the pacemaker lay above the brain stem. He found that if the midbrain of a cat was completely transected, the spontaneous electrical activity that could be recorded from the unanaesthetized cerebrum (cerveau isole) was dominated by slow waves. Dempsey and Morison et al reported a highly original series of investigations in which the non-specific thalamic nuclei were identified as the probable pacemaker region for the EEG. In their initial study these workers stimulated the central end of the cut sciatic nerve in the cat and recorded an early response in the primary somatosensory (SI) cortical area followed by a secondary (non-specific) response which was widely distributed over both hemispheres. They then made lesions in various parts of the thalamus to delineate the neural pathway responsible for the secondary response and were able to establish that the 'specific' somatosensory thalamic nucleus (VPL) was not involved.

Dempsey and Morison et al found that electrical stimulation of the non-specific thalamic nuclei evoked cortical potentials which had the same form and distribution as the secondary responses. The amplitude of these responses to successive stimuli fluctuated in a characteristic manner. While the first response was small, subsequent ones became progressively larger; accordingly Dempsey and Morison termed these potentials recruiting responses. If the stimultion was continued the potentials declined in amplitude. This waxing and waning of responses strongly resemble the spontaneous EEG spindle activity that could be recorded in the same animal under barbiturate

anaesthesia. Further more the recruiting responses were large in those regions of cortex displaying the most prominent EEG spindles, the association areas. It was also noted that good recruiting responses could be obtained only when a spindle was about to begin. This last observation, together with the similarities in appearance and cortical distribution of the recruiting responses and EEG spindles, suggested that the two types of activity were using the same neural pathway, that is, the non-specific nuclei. Further evidence suggest that these nuclei were involved in the genesis of EEG activity, like the certebral cortex, they exhibited spontaneous runs of potentials which waxed and waned corresponding to the waves, in cortical spindles[46]. It has been shown by intracellular recording that the waves in these thalamic spindles are alternating excitatory and inhibitory postsynaptic potentials[45].

THALAMO CORTICAL RELAY CENTRE:

Andersen and Sears have suggested a neural circuit which would account for the frequency and for the waxing and waning of the thalamic waves within a thalamic nucleus. In this Circuit a small number of the thalamic neurons projecting to the cortex (thalamcortical relay or TCR cells) develop Exicatatory Post Synaptic Potentials which is stimulated by background synaptic bombardment from other sites. The impulses discharged by TCR cells will activate an inhibitory interneurone. This interneuron then inhibits a relatively large number of TCR cells, which had not developed a previous Exicatatory Post Synaptic Potentials and impulse. All these TCR

cells now become silent during the basic Inhibitory Post Synaptic Potentials evoked by inhibitory interneuron. As the Inhibitory Post Synaptic Potentials decrements the same cells become hyper excitable due to a fundamental biophysical property of nerve membranes (as in post anodal exaltation). Once more the TCR cells, respond to background excitation and develop Excitatory Post Synaptic Potentials. In the next cycle the discharge of resting cell activates an additional postsynaptic inhibitory neuron so that an even larger fraction of the TCR pool is now inhibited. Subsequent all these resting discharge and the process continues each time the number of discharging thalamic cell increases. After a certain number of cycles the thalamic activity declines, possibly because the discharges of the TCR cells being to lose synchronicity.

The length of each cycle in the circuits will be the combined durations of an Exicatatory Post Synaptic Potentials and Inhibitory Post Synaptic Potentials (approximately 100ms). This periodicity corresponds to a frequency of 10 HZ, which is within the alpha range (8-13 HZ). In a computer model of the thalamus, recurrent axonal branches to postsynaptic inhibitory interneurons can generate synchronized activity which waxes and waves. The synchronization of spindle activity between different thalamic nuclei is brought about by distributor cells; these cells have very long axons which extend from one nucleus to another[45].

The impulses set up in the thalamic cells during an Excitatory Post Synaptic Potentials will trigger Excitatory Post Synaptic Potentials in the cortical cells to which they are linked. A large number of thalamic discharge cell will excite a correspondingly large number of cortical neurons and elicit a good sized surface wave It is seen that axons from non specified thalamic nuclei terminate in all layers of cerebral cortex. An arrangement of this kind would cause the apical dendrites of pyramidal cells to depolarize throughout their entire length and would account for the negativity record at the cortical surface during the recruiting responses and EEG spindles. If the Excitatory Post Synaptic Potentials evoked in the deep pyramidal cells are large, they will initiate action potentials and these in turn, will activate cortical inhibitory interneurons through collateral axonal branches. The inhibitory interneurons will then feed postsynaptic inhibition back on to the deep cortical cells. As the Inhibitory Post Synaptic Potentials dies away it will be succeeded by an Exicatatory Post Synaptic Potentials evoked by the next thalamic discharge and this will influence, the thalamic pacemaker. Nevertheless, the returning cortical volleys are not essential for thalamic rhythmicity since thalamic cells still fire repetitively after the cortex has been ablated[47] .

The routes taken by the non-specific thalamic axons to the cortex are largely unknown, for only the anterior ventral nucleus (VA) has so far been shown to have a direct projection. It is possible that impulses from the

remaining non-specific nuclei may relay in VA or in some other subcortical structure before reaching the cortex.

A polysynaptic path of this kind would account for the rather longer latencies of the recruiting responses compared with those of the 'specific' responses. One subcortical region that may be involved in EEG phenomena is the anterior hypothalamus since low frequency stimulation here produces slow cortical waves together with other physiological manifestations of sleep. A second possibility is that the non-specific thalamic nuclei influence the specific nuclei, which in turn exert control over the cortical areas to which they project. Thus, stimulation of non-specific thalamic nuclei evokes alternating EPSPs and IPSPs in specific thalamic nuclei. The importance of the specific nuclei as the link between the non-specific thalamic nuclei and the cortex is particularly stressed by Andersen and Anderson. In addition, these workers have experimental evidence that, on occasion, a specific nucleus may develop an autonomous rhythm, which is then imparted to the cortex and to the remainder of the thalamus. According to Andersen and Anderson et al any part of the thalamus, specific or non-specific, has the capacity to act as a pacemaker.

Normally, alpha or beta spindles can be recorded from the surface of the brain only during relaxation with the eyes closed or during drowsiness or barbiturate anesthesia. If the relaxed subject opens his eyes or undertakes mental activity the alpha rhythm is replaced by fast irregular activity of small

amplitude, that is, the recorded potentials have become desynchronized. A similar change occurs if strong sensory stimulation is given to a drowsy or lightly anaesthetized animal. In each of these situations the transition is associated with physiological evidence of awakening of the person or animal and since the cerebral cortex has presumably become more excitable, the phenomenon is termed ‘activation’ of the EEG. Because of their relationship to ‘consciousness’ the neural mechanisms involved in activation have been the subject of intensive study. He observed that if the rostral brain stem of a cat was transected, the EEG activity of the isolated cerebral hemispheres was characterized by repetitive slow waves. However, if the transection was made at the junction of the spinal cord and medulla the cortical activity was desynchronized. Clearly some structure in the brianstem was necessary for activiation of the cortex. Magoun and his colleagues identified the activating region with the reticular formation they showed that, in an otherwise intact cat, a lesion in the reticular formation caused the animal to become somnolent and to develop rhythmic cortical activity. In distinction to this finding, a lesion made outside, the reticular formation, so as to interrupt the important lemniscal somatosensory pathways, did not have this retarding effect on the animal’s behaviour nor did it cause synchronization of the EEG. In a different type of experiment Moruzzi and Magoun found that stimulation of the reticular formation produced activation of the EEG under light barbiturate anaesthesia. These crucial experiments have been confirmed and it appears

that the activating region is situated in the mesencephalic part of the reticular formation; in contrast, stimulation of the bulbar or lower pontine regions produces synchronization of the EEG. Since the reticular formation receives inputs from all the sensory pathways, almost any form of powerful stimulation will activate the EEG.

The pathway taken by the reticular axons involved in activation is not altogether clear although many apparently terminate in the non-specific thalamic nuclei. Within the thalamus the reticular fibres evoke excitatory postsynaptic potentials and block inhibitory ones. Consequently there is desynchronization of the thalamocortical relay cells and hence of the cortex itself. Other reticular fibres run to the hypothalamus and it is therefore of considerable interest that lesion of the posterior hypothalamus are associated with hypersomnia; while stimulation of this area produces activation [49].

ALPHA WAVES

Alpha waves generally are seen in all age groups but are most common in adults.

- They occur rhythmically on both sides of the head but are often slightly higher in amplitude on the nondominant side, especially in right-handed individuals.

- They tend to be present posteriorly more than anteriorly and are especially prominent with closed eyes and with relaxation.

- Alpha activity disappears normally with attention (eg, mental arithmetic, stress, opening eyes).
- In most instances, it is regarded as a normal waveform.
- An abnormal exception is alpha coma, most often caused by hypoxic ischemic encephalopathy due to destructive processes in the pons (eg, intracerebral hemorrhage).
- In alpha coma, alpha waves are distributed uniformly both anteriorly and posteriorly in patients who are unresponsive to stimuli.

Beta waves

- Beta waves are observed in all age groups.
- They tend to be small in amplitude and usually are symmetric and more evident anteriorly.
- Many drugs, such as barbiturates and benzodiazepines, augment beta waves.

Theta waves

- Theta waves normally are seen in sleep at any age. In awake adults, these waves are abnormal if they occur in excess.
- Theta and delta waves are known collectively as slow waves.

Delta waves

- These slow waves have a frequency of 3 Hz or less.
- They normally are seen in deep sleep in adults as well as in infants and children.
- Delta waves are abnormal in the awake adult.
- Often, they have the largest amplitude of all waves.
- Delta waves can be focal (local pathology) or diffuse (generalized dysfunction).

K complex

- K complex waves are large-amplitude delta frequency waves, sometimes with a sharp apex.
- They can occur throughout the brain and usually are higher in amplitude and more prominent in the bifrontal regions.
- Usually symmetric, they occur each time the patient is aroused partially from sleep.
- Semiarousal often follows brief noises; with longer sounds, repeated K complexes can occur.

- K complexes sometimes are followed by runs of generalized rhythmic theta waves; the whole complex is termed an “arousal burst”.

V waves

- V waves are sharp waves that occur during sleep. They are largest and most evident at the vertex bilaterally and usually symmetrically.
- They show phase reversal at the vertex.
- V waves tend to occur especially during stage 2 sleep and may be multiple. Often, they occur after sleep disturbances (eg, brief sounds) and, like K complexes, may occur during brief semiarousals.
- V waves are easy to recognize.

Lambda waves

- Lambda waves occur in the occipital regions bilaterally as positive (upgoing) waves.
- They are triangular in shape and generally symmetric.

- They occur in the awake patient and are said to be most evident when the subject stares at a blank, uniform surface.
- Lambda waves occur when reading and occasionally when watching TV.
- Morphologically, they are similar to POSTS both in form and in occipital distribution.

Positive occipital sharp transients of sleep

- POSTS are triangular waves that occur in the bilateral occipital regions as positive (upgoing) waves.
- They can be multiple and usually are symmetric.
- POSTS occur in sleeping patients and are said to be most evident in stage 2 of sleep, although they are not uncommon in stage 1.
- POSTS are similar if not identical to lambda waves both morphologically and in the occipital distribution.

Sleep spindles

- Spindles are groups of waves that occur during many sleep stages but especially in stage 2.

- They have frequencies in the upper levels of alpha or lower levels of beta.
- Lasting for a second or less, they increase in amplitude initially and then decrease slowly. The waveform resembles a spindle.
- They usually are symmetric and are most obvious in the parasagittal regions.

Mu waves - Wicket rhythm

- Mu waves are runs of rhythmic activity that have a specific shape. They are rounded in one direction with a sharp side in the other direction.
- Frequency is one half of the fast (beta) activity.
- Mu waves disappear with motor acts of the contralateral hand or arm.
- Unlike alpha activity, they are not blocked by eye opening.
- They often are asymmetric. Mu waves are seen best when the cortex is exposed or if bone defects (eg, postsurgical) are present in the skull.

- They tend to be more evident over the motor cortex and in the parasagittal regions.

Spikes and sharp waves

- These are recognized by their height, their sharp top, and their narrow base.
- Spikes and sharp waves usually are abnormal.
- They can be normal in the following settings:
- V waves of sleep in the parasagittal regions in stage 2 sleep can be normal.
- Small, sharp spikes of sleep or benign epileptiform transients of sleep (BETS) are nonpathologic. They occur in the temporal regions, often switching from side to side. They do not have slow-following waves as do most of the pathologic spikes of epilepsy.
- Numerous artifacts resemble spikes, but they are distinguished by other waves that may be present, by observation of the patient while they are occurring, and by experience.
- POSTS can have a sharp contour yet be quite normal. They occur in the occipital regions bilaterally during sleep.

Benign epileptic transients of sleep

- These sharp, usually small waves occur on one or both sides (usually asynchronously), especially in the temporal and frontal regions.
- BETS are rare in children but are more frequent in adults and elderly persons.
- Although they can occur in epileptic patients, BETS often are seen in individuals without epilepsy and can be regarded as a probable normal variant.

Slow activity

Abnormal slow activity is by far the most common EEG manifestation of focal brain dysfunction. The abnormality that correlates best with the presence of a structural lesion is polymorphic or arrhythmic (as opposed to monomorphic or rhythmic) delta (ie, 1-3 Hz) slowing. This is all the more reliable when it is continuous, unreactive (ie, characterized by lack of change between states, such as wake or sleep, or in response to external stimuli), of high amplitude, polymorphic, and unilateral. The localization of slow potentials follows the same rules as that of epileptiform discharges. Thus, “phase reversals” are useful to localize slow potentials and do not imply abnormality or epileptogenicity.

AMPLITUDE ASYMMETRY

In the classification used here, the term asymmetry refers to asymmetry of amplitude and to normal rhythms. By contrast, a focal frequency asymmetry would be classified as focal slowing. The amplitude asymmetries should be evaluated on referential montages, since amplitude is highly dependent on interelectrode distances.

Periodic lateralized epileptiform discharges

Described in 1964 by Chatrian et al, periodic lateralized epileptiform discharges (PLEDS) are a special type of focal abnormality. As implied by their name, they are periodic, lateralized, and epileptiform. Periodicity is the most characteristic feature, and the one that sets PLEDS apart from other focal abnormalities. Periodicity refers to a relatively constant interval between discharges, which varies between 0.5 and 3 seconds and most often is around 1 second. The epileptiform morphology of the discharges is not invariable, as PLEDS are often closer to slow waves than to sharp waves in morphology.

SLOW ACTIVITY

Continuous focal slow activity is the only nonepileptiform focal abnormality that can be interpreted unequivocally as abnormal when it is an isolated finding. Other focal abnormalities are quite frequent but are of such low specificity that they almost never constitute an abnormality in themselves.

To be interpreted as abnormal, these usually require the coexistence of a more definite abnormality such as slowing of epileptiform discharges.

As already outlined, focal slowing is nonspecific as to etiology, and in the era of neuroimaging the EEG has no role in diagnosing the nature of a lesion. Focal slowing is the most common abnormality associated with focal lesions of any type, including (but not limited to) neoplastic, vascular, subdural collections, traumatic, and infectious **).** It occasionally may be seen even in more subtle structural abnormalities such as mesiotemporal sclerosis or focal malformations of cortical development.

The physiologic basis for focal polymorphic delta activity caused by focal cortical lesions is not fully understood. It is probably due to abnormalities in the underlying white matter rather than the cortex itself. When present, focal slow activity correlates highly with the side of the lesion, but it is not reliable for lobar localization. The likelihood of a structural lesion (ie, specificity) diminishes, when the slow activity lacks these characteristics and is intermittent, in the theta rather than the delta range, and of low amplitude. This type of slowing may be normal (eg, temporal slowing of the elderly;). This is essentially the difference between focal “continuous slow” and “intermittent slow”

In a few situations in clinical neurology, the EEG may show clear evidence of focal dysfunction (ie, focal slow) while no structural

abnormality is found. The typical cases in point are the focal epilepsies. A readily demonstrable structural lesion usually is not found on neuroimaging, typically MRI .

Focal brain dysfunction without structural abnormalities has been observed in transient ischemic attacks (TIA), migraine, and postictal states. Polymorphic delta activity in these cases may be indistinguishable from that caused by a structural lesion, except that it is short-lived (ie, it disappears over time). The postictal state is the most common cause of nonstructural polymorphic delta activity, but the activity disappears within minutes to hours after the ictal event. Patients with ongoing TIAs or migrain rarely undergo an EEG during the symptomatic period, so clinical data are scarce.

Amplitude asymmetry

Destructive lesions clearly can attenuate the amplitude of normal rhythms. However, normal rhythms are never perfectly symmetric in amplitude, therefore which asymmetries to consider significant is not always clear. Some have proposed a greater than 50% side-to-side difference as abnormal.

A good rule of thumb is that, with very few exceptions, significant focal asymmetries are associated with slowing. Any amplitude asymmetry associated with slowing of frequency be considered significant.

Amplitude asymmetry or suppression of normal rhythms is somewhat more likely to be seen in structural abnormalities that increase the distance or interfere with the conduction of the electrical signal between the cortex and the recording scalp electrodes. Examples include subdural collections (eg, hematoma, empyema), epidural collections (eg, hematoma, abscess), subgaleal collections, and calcifications such as those seen in Sturge-Weber syndrome.

Amplitude asymmetry also may be more common than slowing in subdural hematomas. However, caution must be exercised before considering isolated non epileptiform focal findings other than slowing as abnormal. In general, as with other types of focal EEG abnormalities such as slowing, amplitude asymmetry is nonspecific as to etiology.

Although asymmetry in amplitude is usually indicative of dysfunction on the side of depressed amplitude, one notable exception to this rule is the so-called “breach rhythm”. This is caused by a skull defect, which attenuates the high-frequency filter function of the intact skull. As a result, faster frequencies (eg, alpha, spindles, beta) are of higher amplitude on the side of the defect. Since morphology often is sharply contoured, determining the epileptogenicity of these discharges can be extremely difficult, and in this situation erring on the conservative side, by not interpreting them as epileptiform, is clearly preferable. Because of a cancellation effect between frontopolar (Fp1/Fp2) and frontal (F3/F4), eye movements often they are not

increased on the side of a skull defect and may indeed be of lesser amplitude on that side.

PERIODIC LATERALIZED EPILEPTIFORM DISCHARGES

PLEDS are caused by acute destructive focal lesions and are a transitory phenomenon: they tend to disappear in weeks, even if the causal lesion persists. Over time, the record takes on a less specific focal slow appearance, which is more likely to persist. By far the most common etiology is an acute cerebrovascular event; second most common is focal encephalitis such as that caused by herpes. In a clinical context suggestive of viral encephalitis, the EEG can be of great value for diagnosis and can guide tissue biopsy. Though most often associated with an acute destructive lesion, PLEDS, like other EEG findings, are not specific as to etiology and have been described in almost all types of structural lesions, including subdural hematoma and chronic lesions, especially in the presence of a superimposed systemic disturbance.

In keeping with their epileptiform morphology, PLEDS have a close association with clinical seizures, and on average about 80% of patients with PLEDS have clinical seizures. The transition between PLEDS and a clear ictal seizure pattern is very gradual, illustrating the hypothesis that PLEDS may represent a subclinical ictal pattern. In clinical practice, however, PLEDS usually are managed as interictal discharges (ie, spikes or sharp waves). They

indicate a high risk for focal seizures, but usually are not treated with antiepileptic drugs unless clinical evidence for seizures is noted. This is somewhat controversial, however, and some advocate antiepileptic treatment in all patients with PLEDS.

Periodic patterns in Creutzfeldt-Jakob disease usually are generalized and bisynchronous but occasionally, especially early in the course, they may be unilateral or markedly asymmetric, and thus take on the appearance of PLEDS.

Other less common focal patterns

An abnormal response to photic stimulation can be seen in focal lesions. Normal photic driving has long been known to be potentially reduced on the side of a lesion. Posterior destructive lesions are particularly likely to attenuate the driving response, but some reports have described an enhanced photic response on the side of dysfunction. However, since the normal driving response can be quite asymmetric, such a finding should be accompanied by a more reliable abnormality such as slowing of the waveform frequency in order to be interpreted as abnormal.

The Bancaud phenomenon refers to the unilateral loss of reactivity of a normal rhythm and initially was described in the context of the alpha rhythm. It should be considered a pathological finding only when associated with other more definite abnormalities, such as slowing.

STROKE

BACKGROUND:

Stroke is the clinical term for acute loss of circulation to an area of the brain, resulting in ischemia and a corresponding loss of neurologic function. Classified as either hemorrhagic or ischemic, strokes typically manifest with the sudden onset of focal neurologic deficits, such as weakness, sensory deficit, or difficulties with language. Ischemic strokes have a heterogeneous group of causes, including thrombosis, embolism, and hypoperfusion, whereas hemorrhagic strokes can be either intraparenchymal or subarachnoid hemorrhage.

EPIDEMIOLOGY

They cause 2,00,000 deaths each year and are a major cause of disability. Stroke represents the most common cause of death in developed nations. In India crude prevalence rate for hemiplegia in the range of 200 per 1,00,000 persons nearly 1.5 percent of urban hospital admissions,4.5 percent of all medical and around 20 percent of neurological cases[55].

SEX:

In patients younger than 60 years, the incidence of stroke is greater in males (3:2 ratio).

AGE:

- Stroke can occur in patients of all ages, including children.
- Risk of stroke increases with age, especially in patients older than 64 years, in whom 75% of all strokes occur.

RISK FACTORS

- **NON MODIFIABLE RISK** factors include
- Age
- Race
- Sex
- Ethnicity
- History of migraine headaches
- Sickle cell disease
- Fibro muscular dysplasia and heredity.
- **MODIFIABLE RISK FACTORS** include the following:
    - Hypertension (the most important)
    - Cardiac disease - Atrial fibrillation, valvular disease, mitral stenosis, structural anomalies allowing right to left shunting,

such as a patent foramen ovale, atrial and ventricular enlargement.

- Diabetes mellitus
- Hypercholesterolemia
- Transient ischemic attacks (TIAs)
- Carotid stenosis
- Hyperhomocystinemia
- Lifestyle issues - Excessive alcohol intake, tobacco use, illicit drug use, obesity, physical inactivity.
- Oral contraceptive use

PATHOPHYSIOLOGY:

The brain is the most metabolically active tissue in the body. While representing only 2% of the body's mass, it requires 15-20% of the total resting cardiac output to provide the necessary glucose and oxygen for its metabolism. Ischemic strokes result from events that limit or stop blood flow, such as embolism, thrombosis in situ, or relative hypoperfusion. As blood flow decreases, neurons cease functioning, and irreversible neuronal ischemia and injury begin at blood flow rates of less than 18 mL/100 mg/min.

Majority of thrombotic occlusion are due to atherosclerosis. The most common site of involvement are the carotid bifurcation, the origin of middle cerebral artery. Another important aspect is its frequent association with hypertension, diabetes mellitus, atherosclerotic heart disease, hypercholestrolemia and gout.[40]

Tissue surrounding the core of infarction is ischemic and is referred to as the ischemic penumbra, which can be imagined by perfusion-diffusion imaging using MRI.[41]

The ischemic penumbra will eventually infarct if no change in blood flow occurs. Neuronal cell death occurs by two different pathways:

NECROTIC PATHWAY

Ischemia produces necrosis by starving neurons of glucose which in turn causes failure of mitochondria to produce ATP. Without ATP membrane ion pump stops functioning and neurons depolarise, allowing intracellular calcium to rise. Cellular depolarisation also causes glutamate release from synaptic terminals; excess glutamate produces neurotoxicity by agonising the post synaptic glutamate receptors that increase neuronal calcium influx. Free radicals are produced by membrane lipid degradation and mitochondrial dysfunction.[53]

APOPTOTIC PATHWAY

Ischemic penumbra favours apoptotic cellular death causing cells to die days to weeks later. [53]

PATHOLOGIC FEATURES

a. Cerebral Infarction – Pathologic Features

PATHOLOGICAL CHANGES:

Gross findings

- Acute cerebral infarct : focal swelling and congestion in a well defined vascular territory.
- Subactute: circumscribed regions of congestion, softening, and early cavitation with separation of cortex from the underlying with matter.
- Chronic (remote) infarct : complete cavitation with sparing of the subpial layer of cortex.

Microscopic Findings

- Acute changes (12 to 24 hours after significant hypoxiaschemia) : tissue pallor with acute neuronal necrosis eosinophilic (ischemic) cell change.
- Sub acute changes (2 days to 2 weeks): appearance of macrophages, capillary proliferation, and early astrocytic reaction at the lesion edge.

- Chronic changes (months to years): removal of necrotic tissue by macrophages with resulting cavitation (liquefaction necrosis); residual reactive astrocytes from a gliotic scar at the infarct edges.

ISchemic cascade

The processes involved in stroke injury at the cellular level are referred to as the ischemic cascade. Some of the factors thought to result in cell death and dysfunction are listed here, but others are being discovered at a rapid rate. Within seconds to minutes of the loss of glucose and oxygen delivery to neurons, the cellular ischemic cascade begins. This is a complex process that begins with cessation of the electrophysiologic function of the cells. The resultant neuronal and glial injury produces edema in the ensuing hours to days after stroke, causing further injury to the surrounding neuronal tissues.

Ischemic penumbra

An acute vascular occlusion produces heterogeneous regions of ischemia in the dependent vascular territory. The quantity of local blood flow is comprised of any residual flow in the major arterial source and the collateral supply, if any. Regions of the brain without significant flow are referred to collectively as the core, and these cells are presumed to die within minutes of stroke onset. Zones of decreased or marginal perfusion are collectively called the ischemic penumbra. Tissue in the penumbra can remain viable for several

hours because of marginal tissue perfusion, and currently studied pharmacologic interventions for preservation of neuronal tissue target this penumbra.

Administration of t-PA to the patient with an acute stroke allows attempts to establish revascularization, so that cells in the penumbra can be rescued before irreversible injury occurs. Restoring blood flow can mitigate the effects of ischemia only if performed quickly.

MECHANISMS OF STROKE

EMBOLIC STROKES

Emboli may either be of cardiac or arterial origin. Cardiac sources include atrial fibrillation, recent myocardial infarction (1-3% of all acute myocardial infarctions [AMIs]), prosthetic valves, native valvular disease, endocarditis, mural thrombi, dilated cardiomyopathy, or patent foramen ovale allowing passage of venous circulation emboli. Arterial sources are atherothrombolic or cholesterol emboli that develop in the arch of the aorta and in the extracranial arteries (ie, carotid and vertebral arteries). Embolic strokes tend to have a sudden onset, and neuroimaging may demonstrate previous infarcts in several vascular territories.

Thrombotic strokes

Thrombotic strokes include large-vessel strokes (70%) and small-vessel or lacunar strokes (30%). They are due to in situ occlusions, characteristically on atherosclerotic lesions in the carotid, vertebrobasilar, and cerebral arteries, typically proximal to major branches. Thrombogenic factors include injury to and loss of endothelial cells exposing the subendothelium and platelet activation by the subendothelium, activation of the clotting cascade, inhibition of fibrinolysis, and blood stasis.

Thrombotic strokes are thought to originate on ruptured atherosclerotic plaques. Intracranial atherosclerosis may be the cause in patients with widespread atherosclerosis. In other patients, especially younger patients, other causes should be considered, including coagulation disorders (eg, antiphospholipid antibodies, protein C deficiency, protein S deficiency), sickle cell disease, fibromuscular dysplasia, arterial dissections, and vasoconstriction associated with substance abuse.

Lacunar stroke

Lacunar strokes represent 20% of all ischemic strokes. They occur when the penetrating branches of the middle cerebral artery (MCA), the lenticulostriate arteries, or the penetrating branches of the circle of Willis, vertebral artery, or basilar artery become occluded. Causes of lacunar infarcts include microatheroma, lipohyalinosis, fibrinoid necrosis secondary to

hypertension or vasculitis, hyaline arteriosclerosis, and amyloid angiopathy. The great majority are related to hypertension. Of all stroke types, lacunar strokes have the best prognosis.

Watershed infarcts

- These infarcts, also known as border zone infarcts, develop from relative hypoperfusion in the most distal arterial territories and can produce bilateral symptoms.

MATERIALS AND METHODS

SELECTION OF STUDY SUBJECTS

INCLUSION CRITERIA

Patients with acute cerebrovascular accident admitted to Thanjavur medical college Hospital during Jan 2005 – August 2006 were included in the study.

All patients irrespective of age and sex were included.

Patients who present within 24-72 hours of cerebrovascular accident were included in the study.

EXCLUSION CRITERIA

Patients who are uncooperative and unconscious

Patients with prior seizures

Patients with brain stem infarct, cerebellar infarct

METHOD OF STUDY

STUDY PERIOD:

The study was done during the period Jan 2005 – August 2006.

STUDY DESIGN :

Patients admitted with acute onset of focal neurological deficit were admitted within 24-72 hours were selected. Patients belonged to both sexes of all ages .All patients underwent a thorough and detailed general and neurolgical examination.

All other system examination was done in all the patients.

Neurological examination was done with reference to higher function, cranial nerves, motor, sensory system, autonomic functions.

Complete hemogram and urine analysis ,blood sugar ,blood urea , serum creatinine, serum electrolytes were analyzed.

ECG and Chest Radiograph were also taken

CT SCAN BRAIN

CT Scan brain done within 24 to 72 hours of admission and analyzed for

1. The site of the lesion;
2. Depth of lesion
3. Cortical
4. Sub cortical and multiple infarct,

5. Hemorrhage

6. The midline shift; cerebral edema

ELECTRO ENCEPHALOGRAM

An EEG is taken within 24 hours after admission for all patients with cerebrovascular accident and analyzed for the following wave forms,

1. Slow or fast wave.

2. Focal slowing or diffuse slowing;

3. Spike and Sharp wave activity;

4. Preserved background activity;

5. Local or diffuse polymorphic slowing,

6. FIRDA,

7. Epileptiform activity,

8. Involvement of both hemispheres.

RESULTS AND OBSERVATIONS

RESULTS AND OBSERVATION

AGE AND SEX DISTRIBUTION

The total number of patients in this study was 35. The study was done during the period of Jan 2005– August 2006 in Thanjavur medical college hospital. Out of which 19 were male patients and 16 were female patients.

TABLE 1

AGE / SEX	< 40	41 - 50	51-60	61-70	>70	TOTAL
MALE	2 (5.71)	3 (8.57)	8 (22.86)	5 (14.29)	1 (2.86)	19
FEMALE	2 (5.71)	6 (17.14)	4 (11.43)	3 (8.570	1 (2.86)	16
TOTAL	4 (11.43)	9 (25.71)	12 (34.29)	8 (22.86)	2 (5.71)	35

* Numbers with in the brackets represent percentage.

The total number of male patients included in the study was 19.Out of which 2 patients belong to the age group of less than 40 years. The youngest of them was 34 years old. 3 patients belong to the age group of 41 to 50 years. 8 patients belong to the age group of 51 to 60 years.5 patients belong

to the age group of 61 to 70 years. 1 patient belongs to the age group of above 70 years. The oldest patient in this study was 75 years old.

The number of female patients in the study was 16.Out of which 2 patients belong to the age group of less than 40 years;6 patients belong to the age group of 41 to 50 years;4 patients belong to the age group of 51 to 60 years;3 patients belong to the age group of 61-70, one patient was above the age of 70. The youngest of them was 30 years old and the oldest of them was 70 years old.

In this study cerebrovascular accident occurred commonly in the age group of 50 to 60 years in males. In females it occurred commonly in the age group of 40 to 50 years.

ASSESMENT OF RISK FACTORS

TABLE NO. 2

SEX	Male	Female
Diabetes Mellitus	7 (36.84)	2 (12.5)
Hypertension	15 (78.9)	9 (56.2)
CAHD	7 (36.84)	2(12.5)
Rheumatic heart disease	-	3 (18.75)
Smoker	8 (42.10)	-
Alcoholic	7 (36.84)	-

❖ Numbers within the parenthesis indicate %

In this study 78.9 percent of male patients had hypertension.36.8 percent of male patients had coronary heart disease; 42.10 percent of males are smokers;36.84 percent of male patients are alcoholic; 36.8 percent of male patients are having diabetes mellitus.

In this study 12.5 percent of females patients have diabetes mellitus;56.2 percent of female patients have hypertension;12.5 percent of female patients have coronary heart disease;18.75 percent of female patients have rheumatic heart disease.

In this study most common risk factor associated with cerebrovascular Accident is Hypertension followed by smoking.

In the females hypertension is commonest risk factor associated with cerebrovascular accident.

2 patients had Rheumatic heart disease, Mitral Stenosis Mitral regurgitation, Pulmonary Hypertension. 1 Patient had Rheumatic heart disease mitral stenosis, Pulmonary Hypertension.

CT SCAN BRAIN

TABLE : 3

S.No	TYPE OF LESION	MALE	%	FEMALE	%
1.	CORTICAL INFARCT	6	31.58	2	12.5
2.	SUBCORTICAL INFARCT	1	5.26	4	25
3.	MULTIPLE INFARCT	9	47.37	5	31.25
4.	NORMAL	3	15.79	5	31.25
TOTAL		**19**		**16**	

Out of 35 study patients 6 male patients had cortical infarct; 1 male patient had sub cortical infarct. 9 male patients had multiple infarct; and 3 male patients had normal CT scans.

Among the 16 female patients 2 female patients had cortical infarct; and 4 female patients had sub cortical infarct. 5 female patients had multiple infarct.5 female patients had normal CT scan findings. In this study multiple infarct was common among males than females.

EEG FINDINGS

TABLE : 4

S.No	ELECTROENCEPHALOGRAPHY	MALE	FEMALE
1.	FAST BACKGROUND	1	0
2.	SLOW BACKGROUND	3	3
3.	FOCAL SLOWING	5	2
4.	DIFFUSE SLOWING	1	0
5.	FOCAL POLYMORPHIC	2	0
6.	DIFFUSE POLYMORPHIC	1	0
7.	FIRDA	2	0
8.	SPIKE AND SHARP WAVE	2	1
9.	EPILEPTIFORM	1	1
10.	NORMAL	7	11

Out of 35 patients, 5 male patients had focal slowing; and 2 female patients had focal slowing. 1 male patient had diffuse slowing . 1 male patient had fast background. 3 male patient had slow background and 3 female patients had slow background. 2 male patient had focal polymorphic slowing . 1 male patient had diffuse polymorphic slowing. 2 male patient had focal intermittent rhythmic delta activity. 2 male patient had spike and sharp wave pattern.1 female patient had spike and sharp wave pattern. 1 male patient had epileptiform activity;1 female had epileptiform activity. 7 male patients had normal EEG and 11 female patients had normal EEG

CT SCAN AND EEG CORRELATION

TABLE :5

S.No	SEX	CT Scan		EEG	
		Normal	Abnormal	Normal	Abnormal
1.	MALE	3	16	8	11
2.	FEMALE	5	11	11	5

Among the 19 male patients CT Scan was normal in 3 patients ;16 male patients had abnormal CT Scan.

Among the 19 male patients EEG was normal in 8 patients; and 11 male patients had abnormal EEG.

Among the 16 female patients 5 female patients had normal CT Scan; and 11 female patients had abnormal CT Scan.

Among the 16 female patients EEG was normal in 11 patients and 5 female patients had abnormal EEG.

NORMAL CTSCAN AND ABNORMAL EEG

TABLE NO. : 6

Sex	Normal CT Scan	Abnormal EEG
Male	3	3
Female	1	1

Out of 19 male patients. 3 male patients had normal CT Scan and an abnormal EEG. Out of 16 female study patients 1 female patients had normal CT Scan and an abnormal EEG.

In 2 male patients with left hemi paresis CT Scan was normal. But EEG showed focal slowing in the right fronto parietal region. In one male patient with right hemi paresis the CT Scan was normal. But the EEG showed bilateral focal slowing more on right side with intermittent sharp wave activity. In one female patient with right hemiparesis CT Scan was normal and EEG showed bilateral slow wave more on the left side.

ABNORMAL CT SCAN AND NORMAL EEG

TABLE NO. 7

Sex	Abnormal CT	Normal EEG
Male	7	7
Female	7	7

Out of 19 study patients 7 male patients had normal EEG and abnormal CT Scan. Out of 16 Female study patients 7 female patients had normal EEG an abnormal CT Scan.

Out of 7 male patients, 3 male patients with left hemiparesis CT scan showed Right subcortical infarct and EEG was normal. 1 male patient with right hemiparesis CT scan showed left cortical infarct and EEG was normal. 3 male patients with right hemiparesis, CT scan showed bilateral subcortical infarct but the EEG was normal.

Out of 7 female patients, 2 female patients with right hemiparesis had normal EEG and CT scan showed left sub cortical infarct. 1 female patient with right hemiparesis had normal EEG and CT scan showed left cortical infarct. 1 female patient with left hemiparesis EEG was normal and CT scan showed right sub cortical infarct. In 2 female patients with left hemiparesis, EEG was normal and CT scan showed multiple infarct. 1 patient with right hemiparesis had multiple infarct.

ABNORMAL EEG AND ABNORMAL CT SCAN

TABLE NO. 8

Sex	Abnormal EEG	Abnormal CT Scan
Male	9	9
Female	4	4

Out of 19 male patients 9 male patients had abnormal EEG an abnormal CT Scan. Out of 16 female study patients 4 female patients had abnormal EEG and abnormal CT Scan.

In our study out of 9 male patients with abnormal EEG and abnormal CT scan, 4 patients with left hemiparesis had right cortical infarct in the CT scan. Among these 4 patients 3 patients had focal slowing, and one patients with Right cortical infarct had diffuse slowing. 1 out of 9 had right hemiparesis and left cortical infarct and EEG showed bilateral FIRDA with intermittent spike and sharp wave activity and bilateral epileptiform activity.

1 out of 9 male patients with right hemiparesis CT scan showed multiple infarct and EEG showed slow waves in left fronto temporal region.

3 out of 9 male patients with left hemiparesis, CT scan showed multiple infarct and EEG showed in 2 out of these 3 patients had slow wave

right side and one patient had bilateral FIRDA with diffuse polymorphic slowing.

Out of 4 female patients one patient with left hemiparesis CT scan showed multiple infarct and EEG showed focal slowing right centro parietal region. One patient with left hemiparesis, CT scan showed sub cortical infarct and EEG showed right fronto parietal slowing. In one patient with right hemiparesis CT scan showed multiple infarct and EEG showed slow wave on the left fronto parietal region. In one female patient with left hemiparesis CT scan showed right cortical infarct and EEG showed right fronto parietal slowing with epileptiform activity.

NORMAL EEG AND NORMAL CTSCAN

TABLE NO. 9

Sex	Normal EEG	Normal CT
Male	0	0
Female	3	3

Out of 35 Study patients no male patient had normal EEG an normal CT Scan. Out of 35 study patients 3 female patients had normal EEG an normal CT Scan.

Out of 3 patients with normal EEG and normal CT scan, 2 patients had left hemiparesis and 1 patient had right hemiparesis.

EEG IN CORTICAL AND SUBCORTICAL INFARCT AND MULTIPLE INFARCT

TABLE 10

EEG	Cortical infarct		Sub cortical infarct		Multiple Infarct	
	Normal	Abnormal	Normal	Abnormal	Normal	Abnormal
Male	1	5	1	0	5	4
Female	1	1	3	1	3	2

Out of 19 male patients 1 male patients with cortical infarct had normal EEG and 5 male patients with cortical infarct had abnormal EEG.

Out of 19 male patients male patients with sub cortical infarct 1 male patient had normal EEG.

Out of 19 male patients 5 patients with multiple infarct had normal EEG and 4 patients with multiple infarct had abnormal EEG.

Out of 16 female study patients 1 female patients with cortical infarct had normal EEG and 1 female patients with cortical infarct had abnormal EEG.

Out of 16 female study patients 3 female patients with sub cortical infarct had normal EEG and 1 female patients with sub cortical infarct had abnormal EEG. Out of 16 female patients with multiple infarct 3 female patients had normal EEG and 2 patient had abnormal EEG.

DISCUSSION

All the 35 patients selected for this study were thoroughly examined and the diagnosis of cerebrovascular accident was made. EEG and CT Scan brain were done for all these patients.

AGE AND SEX INCIDENCE

In this study among males cerebrovascular accident occured commonly in the age group of 50 to 60 years.

In females cerebrovascular accident occured commonly in the age group of 40 to 50 years.

Two male and female patients had young stroke.

RISK FACTORS

Out of 19 male patients included in this study ,stroke occured commonly in patients with hypertension. The other risk factors present in this study were diabetes mellitus, coronary heart disease ,smoking and alcoholism.

Collins and associates proved that long term control of hypertension decreases the incidence of both infarction and intra cerebral hemorrhage.[51]

Most common cause of embolic stroke is structural heart disease and atrial fibrillation [50]

In this study 3 female patients had stroke associated with rheumatic heart disease. Out of which two patients had multiple infarct and one patient had normal CT Scan .The ECHO showed left atrial clot in 2 patients with multiple infarct. In 1 patient there was no left atrial clot

CT SCAN FINDINGS

In this study, CT Scan was able to detect infarct in 27 cases of focal neurological deficit.

JANATI A ,BALACHANDRAN S, and his coworkers studied 50 patients with stroke. In their study 4 patients had normal CT Scan but the EEG was abnormal .In our study, out of 35 patients 8 patients had normal CT Scan and among 8 patients EEG was abnormal in 4 cases. This is because CT Scan done within 24 hours after an infarction generally shows no abnormality .In acute stroke the infarct may not be visible for 24–48 hours .Small infarct in cortical surface may also be missed.

In acute cerebral infarction cellular edema occurs .This cellular edema produces a change in the tissue density. These changes are subtle in the early stage of stroke. This is the main limitation of CT Scan .In 30 to 50 percent of patients CT Scan shows no abnormality.

Hence in patients with normal CT Scan we have to do MRI imaging of brain. MRI is costlier and is not available in all medical centers. In this situation EEG can be used to detect infarct and be used to manage the patients.

EEG FINDINGS

In this study EEG was abnormal in 16 patients with infarct and was normal in 19 patients with infarct. In a study conducted by *Van der drift* ,in patients with subcortical infarct EEG showed no or little focal slowing .

Out of 5 patients with subcortical infarct, 1 patients EEG showed fronto parietal slowing. This is due to relative ischemia in superficial branches of middle cerebral artery. In 4 patients with subcortical infarct EEG was normal due to the cortical garland of normal tissue.

In a study conducted by *MURRI. L. GORI. S. et al* the EEG revealed abnormalities in 40 out of 55 cases with infarct. In our study EEG detected abnormalities in 16 out of 35 cases.

In a study conducted by *AHMED et al* abnormal background activity contralateral to the lesion side was associated with alteration of consciousness.

In our study two patients had abnormal background activity contralateral to lesion side and was not associated with unconsciousness.

In a study conducted by *RASHEVA et al,* spikes and sharp waves was seen in 57 percent of patients with embolic stroke. In our study 3 patients had spike and sharp wave activity.

In the study done by *JABBARI et al*, preserved background activity indicates good prognosis .In our study background activity was preserved in 25 patients.

EEG AND CT SCAN FINDINGS CORRELATION

EEG is useful in patients with acute stroke and normal CT scan. If the initial CT Scan excludes hemorrhage and does not detect infarct , EEG can be done to detect infarct.

A. NORMAL CT SCAN AND ABNORMAL EEG

In a study conducted by *FISCH BJ PEDLEY et al* EEG showed focal abnormalities in the absence of CT Scan finding. In our study 3 male and one female patient had normal CT scan and abnormal EEG.

Among these 3 male patients EEG showed focal slowing in right fronto parietal region in two male patients with left hemiparesis.

EEG showed bilateral focal slowing more on right fronto parietal region in one male patient with right hemiparesis.

EEG showed bilateral slow waves more on the left fronto

parietal region in one female patient with right hemiparesis.

From the above study ,it is observed that EEG can be used to detect infarct in patients with acute stroke; particularly if the initial CT Scan is normal.

B. NORMAL CT SCAN AND NORMAL EEG

In 3 patients both CT Scan and EEG were normal. In these patients MRI brain may help to detect infarct. It should be noted at this juncture that both CT Scan EEG are not 100% sensitive and 100 % specific.

C. ABNORMAL CT SCAN AND NORMAL EEG

In 14 patients with abnormal CT Scan EEG was normal. Among 14 patients 6 patients had subcortical infarct and EEG was normal. In two patients with cortical infarct EEG was normal. In six patients with multiple infarct EEG was normal.

D. ABNORMAL CT SCAN AND ABNORMAL EEG

In 4 patients with cortical infarct, EEG showed focal slowing in the corresponding region. Only one patient with right cortical infarct had diffuse slowing. In one male patient with left cortical infarct EEG showed bilateral FIRDA and bilateral epileptiform activity.

Out of 8 patients with cortical infarct EEG was normal in 2 patients. These two patients had small parietal infarct, which was missed by EEG.

In one male patient with left cortical infarct EEG showed bilateral FIRDA and epileptiform activity. Thereby EEG can be used to detect bilateral changes.

In one male patient with Multiple infarct EEG showed bilateral Frontal Intermittent Rhythmic Delta Activity (FIRDA).

In one patient there was fast background activity .Fast background activity indicates that neurons are surviving in the zone of ischemia[13, 14]. This indicates good prognosis. Thrombolytic therapy will be useful in these patients.

CONCLUSION

CONCLUSION

Role of EEG in patients with CVA

1. This study shows that EEG is useful in detecting the infarct in a group of patients with normal CT Scan and acute stroke. Hence active intervention can be started to prevent the death of neuron.

2. This study also shows that EEG cannot be used as a sole diagnostic tool in patients with cerebrovascular Accident but it is useful when the EEG in used along with CT Scan.

3. EEG can be used in patients where CT Scan is normal and MRI facilities are not available.

This study also shows that in future, EEG may be used for continuous monitoring [25]. In patients with acute cerebrovascular accident it can also be used to assess the severity of stroke.

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PROFORMA

NAME : AGE : SEX :

IP No. : WARD : UNIT :

Date of Admission :

Presenting Complaints :

Past History :

RISK FACTORS :

Diabetes Mellitus :

Hypertension :

Coronary Heart Disease :

Atrial Fibrillation :

Rheumatic Heart Disease :

Smoker :

Alcoholic :

GENERAL EXAMINATION :

Pallor cyanosis Icterus

Clubbing pedal edema Generalized lymphadenopathy

Carotid Bruit

VITALS Temperature

Pulse Rate

Blood Pressure

Respiratory Rate

Central Neruous System :

Higher Mental Function

Cranial Nerves

Spinomotor System :

Sensory system :

Cerebellar system :

Autonomic Nervous system :

Cardio vascular system :

Respiratory system :

Abdominal system :

Investigation

HB : TC : DC : Urine – albumin
- Sugar

B Sugar : B. Urea : S. Creatinine :

S. Sodium : S. Potassium :

ECG :

CHEST RADIOGRAPH :

CT Brain

Cortical Infarct

Sub cortical Infarct

Multiple Infarct

Midline Shift / Brain Edema

Hemorrhage

EEG :

S. No.	IP Number	Name	Age	Sex	Clinical Presenta-tion	Risk Factors	B.P. mm/ Hg	ECG	B.Sugar	B. Urea	S creat	Se. Na^+	Se. K^+	C.T. Scan	EEG	ECHO
1.	912099	Rajendiran	45	M	Lt Hemi paresis	CAH D	130/80	QS V1-V4	108	25	0.8	131	3.9	Rt Cortical infarct	Diffuse Slowing Rt parietal region -	-
2.	906051	Ananda Varman	45	M	Lt – Hemi paresis	DM /HT Smoke r	160/100	Normal	195	32	1.0	134	4.2	Rt Cortica l infarct	Focal Slowing Rt Parietal region -	-
3.	904964	Ramasamy	60	M	Rt- Hemi paresis	DM HT	140/80	Normal	140	40	1.2	136	3.5	Lt Cortica l infarct	Bilateral FIRDA with intermittent sharp and spike B/L epileptiform activity	-
4.	904118	Pitchaiyan	60	M	Lt – Hemi paresis	DM HT	150/60	T↓ V1-V6	232	37	1.1	135	4.2	Normal	Focal slowing Rt fronto parietal region	-
5.	904329	Ramasamy	70	M	Rt – hemi paresis	HT	150/100	Normal	75	33	1.0	135	3.5	Multiple infarct	Normal	-

6.	887850	Samikannu	64	M	Lt-Hemi paresis	HT / CAHD Smoker Alcoholic	150/80	Old IWMI	60	48	1.2	119	4.3	Rt Cortical infarct	Focal slowing right parietal region	-
7.	888302	Jaganathan	52	M	Lt – Hemi paresis	Smoker	124/86	Normal	101	55	1.7	135	4.2-	Multiple infarct	Slow Background Rt- side prominent – mixed with α activity focal polymorphic slowing Rt – Occipital region	-
8.	889780	Vaithiya-lingam	60	M	Rt – Hemi paresis	HT CAH D	180/120	Normal	74	40	1.0	135	3.9	Multiple infarct	Normal	-
9.	908674	Mahathma	67	M	Rt – Hemi paresis	HT CAH D	140/90	Lat Wall Ischemia	74	40	1.0	135	3.9	Normal	Bilateral focal slowing more on Right side with intermittent sharp wave activity	-
10.	889376	Santhosam	55	M	Rt Hemi paresis	CAHD Smoker Alcoholic	100/70	Old ASMI	86	30	1.0	135	3.8	Multiple infarct	Slow wave left fronto temporal region	-

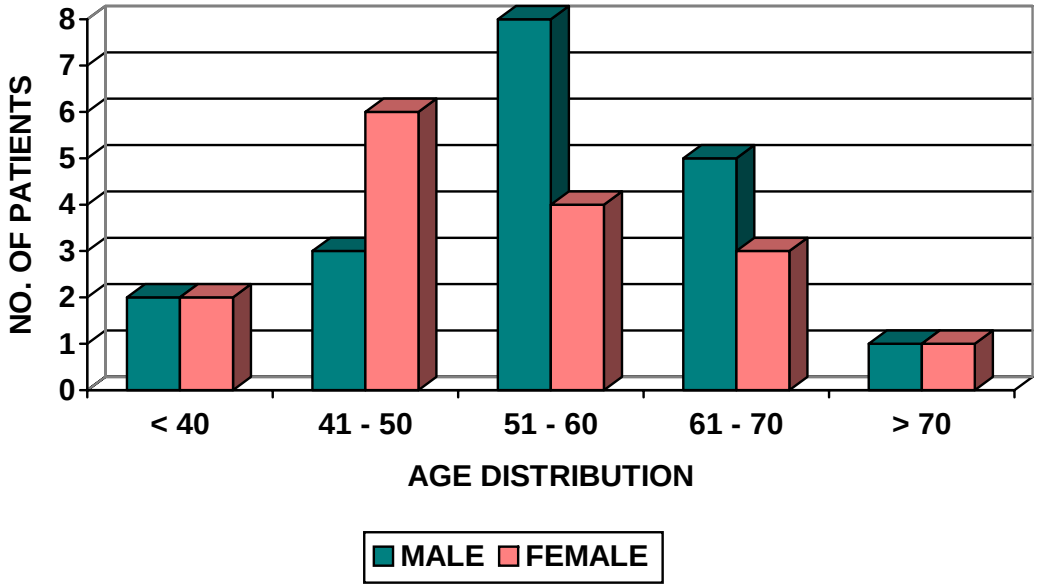
11.	887883	Vivekanandan	35	M	Lt Hemi paresis	HT Alcoholic	130/90	LVH	108	40	1.0	135	3.2	Multiple infarct	Slow Background and Bilateral FIRDA diffuse polymorphic slowing	-
12.	889781	Thulasi Raman	50	M	Rt – hemi paresis	DM HT smoker Alcoholic	140/100	Normal	166	24	0.7	133	3.6	Multiple infarct	Normal	-
13.	889798	Radhakrishnan	34	M	Lt – Hemi paresis	Smoker Alcoholic	140/80	Normal	85	22	0.8	133	3.8	Rt Sub Cortical infarct	Normal	-
14.	900855	Thangavel	67	M	Lt – Hemi paresis	DM / HT CAHD Alcoholic	160/90	Normal	148	46	1.3	135	4.1	Multiple infarct	Polymorphic delta activity over Right fronto parietal region and both frontal regions	-
15.	950504	Antony samy	55	M	Lt – hemi arises	HT	140/80	Normal	127	22	1.0	136	3.8	Multiple infarct	Normal	-
16.	890026	Murugesan	65	M	Lt – Hemi paresis	HT Smoker	170/100	LVH	67	40	1.2	131	4.2	Multiple infarct	Normal	-

17.	904127	Pitchaiyan	60	M	Lt – Hemi paresis	DM/ HT	140/60	Normal	132	38	1.1	135	4.2	Normal	Focal slowing right side	-
18.	888634	Marudhan	60	M	Rt – Hemi paresis	HT Smoker Alcoholic	140/100	Old AWMI	160	28	0.9	136	3.6	Lt Cortical infarct	Normal	-
19.	889034	Natarajan	75	M	Lt – Hemi paresis	HT CAHD	150/90	Lat wall ischemia	85	28	0.8	135	4.2	Rt – Cortical infarct	Focal slowing in Parietal region	-
20.	902355	Sowriyammal	40	F	Rt – Hemi paresis	RHD / MS / PHT	140/100	Normal	91	40	1.2	135	4.3	Multiple infarct	Normal	RHD / MS / PHT
21	901337	Valliyammal	50	F	Rt – Hemi paresis	-	140/100	Normal	118	36	1.0	136	4.0	Left sub cortical infarct	Normal	-
22.	903357	Saithammal	60	F	Lt – Hemi paresis	CAHD HT	140/90	Inf wall ischemia	144	30	0.9	131	4.2	Right sub cortical infarct	Normal	-
23	901338	Nagammal	65	F	Lt- Hemi paresis	-	120/80	Normal	93	36	1.0	132	4.6	Multiple infarct	Right Centro parietal intermittent focal slowing	-

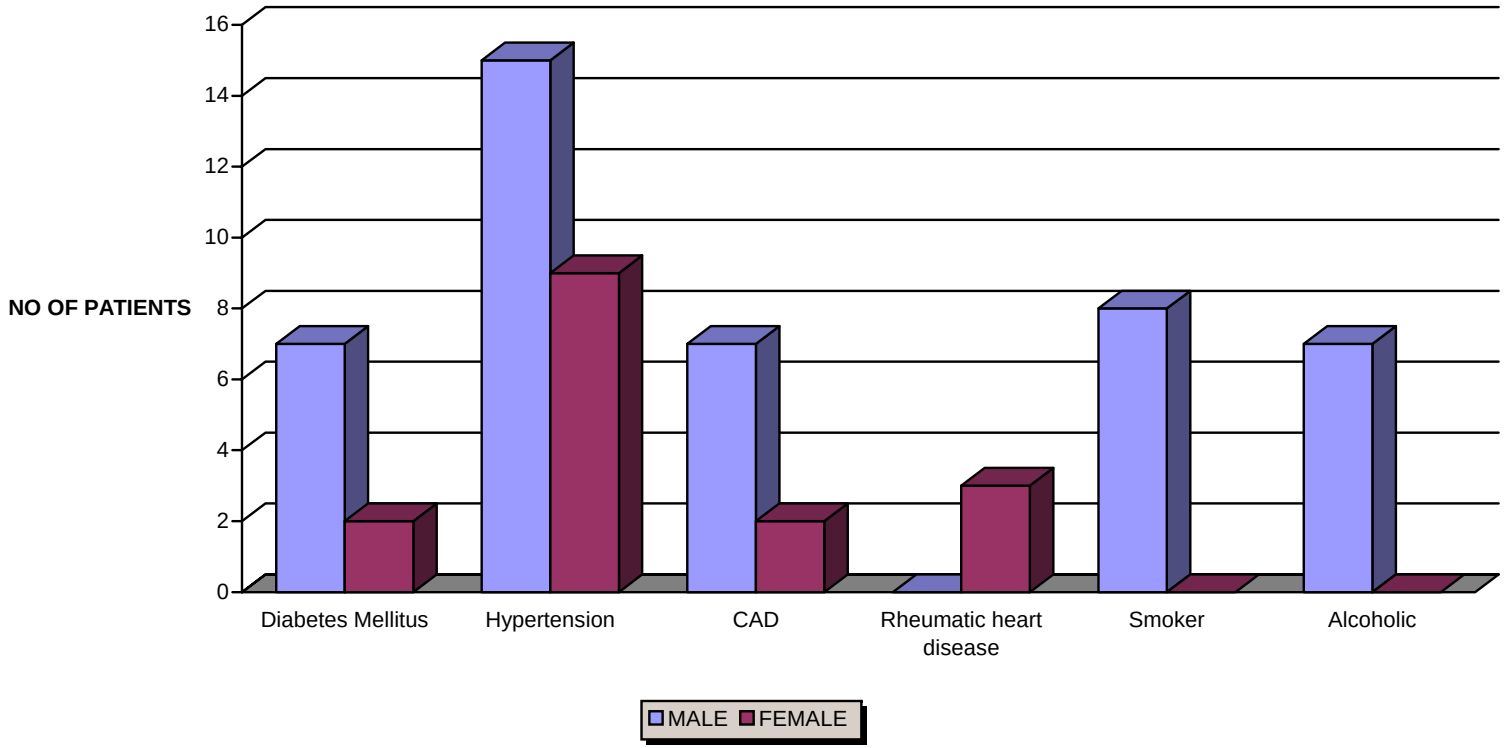
24.	900557	Pushpavalli	70	F	Lt – Hemi paresis	HT	180/90	Normal	107	34	1.2	133	4.4	Rt cortical infarct	Right fronto parietal slowing with epileptiform activity	-
25.	889773	Dharmamal	65	F	Lt – hemi paresis	DM / HT	160/90	Normal	240	33	1.0	138	4.2	Multiple infarct	Normal	-
26.	900674	Muthu lakshmi	50	F	Rt-hemi paresis	HT	140/80	Normal	140	39	1.0	132	4.5	Left sub cortical infarct	Normal	-
27.	912316	Mahalakshmi	49	F	Lt – Hemi paresis	RHD / MS / MR/ PHT AF	130/90	AF	110	2.6	0.7	137	3.9	Multiple infarct	Normal	RHD / MS / MR / PHT / AF / LA CLOT
28.	9111101	Chellammal	62	F	Rt-hemi paresis	-	130/80	Normal	160	30	0.9	138	4.1	Normal	B/l Slow wave more on left side	
29.	912058	Kamatchi	30	F	Rt-Hemi paresis	RHD / MS / MR / PHT / AF	130/80	LAE	54	26	0.6	133	3.7	Normal	Normal	RHD / MS / MR / PHT / AF / LA CLOT

30.	910130	Parvathy	45	F	Rt – Hemi paresis	HT	150/80	Normal	68	28	0.8	136	3.8	Multiple infarct	B/L slow wave more on left fronto parietal region	-
31.	912007	Thenmozhi	45	F	Lt – Hemi paresis	CAH D	110/80	Normal	80	32	0.7	138	3.9	Normal	Normal	-
32.	911079	Samiyammal	60	F	Lt - Hemi paresis	HT	230/90	VPC LVH	104	33	1.0	134	4.0	Normal	Normal	-
33.	908681	Ponnammal	59	F	Rt – Hemi paresis	HT	140/80	Normal	100	31	0.9	133	4.2	Left cortical infarct	Normal	-
34.	904106	Jayapappa	48	F	Lt – Hemi paresis	HT	210/100	Lat wall ischemia	68	52	1.7	133	4.2	Rt-sub cortical infarct	Right Fronto parietal focal slowing	-
35.	905268	Kanthimathi	55	F	Rt-Hemi paresis	DM / HT	160/90	Normal	344	23	0.8	136	3.9	Normal	Normal	-

AGE AND SEX DISTRIBUTION



ASSESSMENT OF RISK FACTORS



CT SCAN BRAIN